

Task Force 5: Systemic Hypertension

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GENERAL CONSIDERATIONS

Systemic hypertension is the most common cardiovascular condition observed in competitive athletes. The diagnosis of hypertension is based on the presence of blood pressure (BP) persistently at or above certain levels as measured by routine sphygmomanometry on at least two separate occasions. A level above 140/90 mm Hg is considered to be hypertensive in people over age 18 years (1). In children and adolescents, hypertension is defined as average systolic or diastolic levels greater than or equal to the 95th percentile for gender, age, and height (2). In determining the level of competitive athletic activity that a hypertensive person may assume, it is also important to ascertain the degree of hypertension-related target organ damage. Although hypertension may be associated with an increased risk for complex ventricular arrhythmias and sudden death, this disease by itself has not been incriminated as a cause of sudden cardiac death in young competitive athletes (3). For the general population, increased levels of noncompetitive physical activity are regarded not only as beneficial by reducing BP (4) and the incidence of hypertension (5), but also protecting against stroke (6). In view of the striking increase in obesity-induced hypertension among children and adolescents related in part to physical inactivity, all people should be encouraged to engage in regular exercise. Those who are hypertensive derive protection from all-cause and cardiovascular mortality by maintaining higher levels of cardiorespiratory fitness (7).

Assessment of blood pressure. Blood pressure should be accurately measured in all individuals who wish to participate in competitive athletics before they begin training. Blood pressure should be measured by routine sphygmomanometry, using the guidelines listed in Table 1 (8). There are often “white-coat” elevations induced by anxiety related to the examination, particularly in young people concerned about the potential consequences of the examination. Therefore, additional BP recordings outside the office should be obtained in those with elevated office readings, either with readily available and inexpensive home self-recorders or with less accessible and more expensive automatic ambulatory monitors.

Evaluation. Those individuals with any degree of persistent hypertension should have a thorough history and physical examination and limited laboratory testing to evaluate secondary causes and to ascertain target organ damage (1). If agents that may raise BP such as non-steroidal anti-inflammatory drugs (NSAIDs) are being taken, additional measurements should be obtained after they have been discontinued. Laboratory testing for most

subjects with stage 1 hypertension (140 to 159 mm Hg/90 to 99 mm Hg) should include an automated blood chemistry (glucose, creatinine, or the corresponding estimated glomerular filtration rate, electrolytes, lipid profile), hematocrit, urine analysis, and an electrocardiogram. If hypertension is stage 2 (greater than or equal to 160/100 mm Hg), if results of the initial laboratory tests are abnormal, or if features suggestive of identifiable (secondary) causes are noted by history or physical examination, the patient should be referred for additional study (including echocardiography) and therapy.

Effects of exercise on BP. As noted during stress testing, systolic BP rises during aerobic (dynamic) exercise. Both systolic and diastolic pressures rise even more acutely and to higher levels during resistance (static or isometric) exercise. Because strenuous aerobic or resistance exertion may precipitate myocardial infarction and sudden death in susceptible, untrained individuals (9), those individuals who wish to engage in competitive athletics should increase exercise levels gradually to avoid such cardiac catastrophes.

However, repetitive performance of both aerobic and resistance exercise lowers systolic and diastolic BP (4,10). After each 30-min period of aerobic exercise at 50% of maximal oxygen uptake, the blood pressure remains lower for up to 24 h, with an even greater reduction after 30 min of aerobic exercise at 75% of maximal oxygen uptake (11). As a consequence, the risks of developing elevated BP (5) and of incurring a cardiovascular consequence of hypertension (6,7) are less in those who maintain higher levels of physical activity. Some conditioned athletes (particularly young men), with a slow heart rate and compensatory increase in stroke volume, have high systolic BP that is considered “spurious” hypertension (12) but should nevertheless be carefully monitored.

In normotensive untrained subjects, an excessive rise in systolic BP to above 200 mm Hg during an exercise stress test is predictive of a greater likelihood of the development of persistent hypertension in the future and may be associated with subtle systolic dysfunction (13) and an increased risk of subsequent cardiovascular disease (14). Therefore, such subjects should be advised to increase levels of physical activity gradually to moderate such excessive rises in pressure. Such rises should not restrict activity in those who are well conditioned. Because intensive resistive training may reduce arterial compliance with potential adverse consequences (15), such training should be limited in those athletes with hypertension.

Effects of blood pressure on exercise. Untreated hypertension in athletes may be accompanied by some limitation in exercise performance (16). Before initiating drug therapy, athletes should be strongly encouraged to adopt healthy

Table 1. Guidelines for Blood Pressure Measurement

Posture

Blood pressure obtained in the seated position is recommended. The subject should sit quietly for 5 min, with the back supported in a chair, with feet on the floor, and the arm supported at the level of the heart, before recording blood pressure.

Circumstances

No caffeine during the hour preceding the reading.
No smoking during the 30 min preceding the reading.
A quiet, warm setting.

Equipment

Cuff size

The bladder should encircle and cover at least 80% of the length of the arm; if it does not, use a larger cuff. If bladder is too short, misleadingly high readings may result.

Manometer

Use a mercury, recently calibrated aneroid, or validated electronic device.

Technique

Number of readings

On each occasion, take at least two readings, separated by as much time as is practical. If readings vary by greater than 5 mm Hg, take additional readings until two consecutive readings are close. If the arm pressure is elevated, take the measurement in one leg (particularly in patients less than 30 years old). Initially, take pressures in both arms; if the pressures differ, use the arm with the higher pressure. If the initial values are elevated, obtain two other sets of readings at least 1 week apart.

Performance

Inflate the bladder quickly to a pressure 20 mm Hg above the systolic pressure, as recognized by the disappearance of the radial pulse.
Deflate the bladder 2 mm Hg per second.
Record the Korotkoff phase I (appearance) and phase V (disappearance).
If the Korotkoff sounds are weak, have the patient raise the arm, open and close the hand 5 to 10 times and then reinflate the bladder quickly.

Recordings

Blood pressure, patient position, and arm and cuff size.

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lifestyle behavior and avoid tobacco in any form, excess alcohol, drugs of abuse (especially sympathomimetics such as cocaine or ephedra), androgens, steroids, growth hormone, NSAIDs, and excessive sodium intake. It should be emphasized that the use of antihypertensive drugs may further limit exercise capacity, more so with beta-adrenergic receptor blocking agents than with vasodilators (alpha-adrenergic blocking agents, angiotensin-converting enzyme inhibitors, angiotensin II-receptor blockers, or calcium channel blockers). Indeed, high-intensity competitive athletes may find it very difficult to perform satisfactorily while using beta-blockers (17).

Recommendations:

1. Before individuals commence training for competitive athletics, they should undergo careful assessment of BP and those with initially high levels (above 140/90 mm Hg) should have out-of-office measurements to exclude isolated office “white-coat” hyper-

tension. Those with pre-hypertension (120/80 mm Hg up to 139/89 mm Hg) should be encouraged to modify lifestyle but should not be restricted from physical activity. Those with sustained hypertension should have echocardiography. Left ventricular hypertrophy (LVH) beyond that seen with “athletes’ heart” should limit participation until BP is normalized by appropriate drug therapy.

2. The presence of stage 1 hypertension in the absence of target organ damage including LVH or concomitant heart disease should not limit the eligibility for any competitive sport. Once having begun a training program, the hypertensive athlete should have BP remeasured every two to four months (or more frequently, if indicated) to monitor the impact of exercise.
3. Athletes with more severe hypertension (stage 2), even without evidence of target organ damage such as LVH, should be restricted, particularly from high static sports (classes IIIA to IIIC), until their hypertension is controlled by either lifestyle modification or drug therapy.
4. All drugs being taken must be registered with appropriate governing bodies to obtain a therapeutic exemption.
5. When hypertension coexists with another cardiovascular disease, eligibility for participation in competitive athletics is usually based on the type and severity of the associated condition.

doi:10.1016/j.jacc.2005.02.012

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Appendix 1. Author Relationships With Industry and Others

Name	Research Grant	Scientific Advisory Board	Speakers' Bureau	Steering Committee
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Task Force 6: Coronary Artery Disease

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ATHEROSCLEROTIC CORONARY ARTERY DISEASE

General considerations. Compelling evidence indicates that physical activity reduces cardiovascular events in healthy subjects and cardiac mortality in patients with diagnosed coronary artery disease (CAD) (1). Despite these beneficial exercise effects, vigorous physical activity also transiently increases the risk of both acute myocardial infarction (AMI) (2–4) and sudden cardiac death (SCD) (5–7) with the greatest exercise risk among the most habitually sedentary individuals (2,4,7).

Atherosclerotic CAD is the most frequent cause of these exercise-related cardiac events in adults (8), variously defined as

older than 30, 35, or 40 years of age. Both plaque rupture (9,10) and possibly plaque erosion (11) have been implicated as the immediate cause of exercise-related events in adults, although plaque rupture is more frequent. Several studies over the last decade document that cardiac events frequently occur in coronary arteries that were not previously critically narrowed. This appears to be particularly true for exercise-related cardiac events because angiographic studies of exercise-related AMI in the general population (4) and in sport participants (10) demonstrate less extensive CAD than in comparison subjects. This observation may reflect either selection bias for less severe atherosclerosis in those capable of exercising at high intensity